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(FILE 'HOME' ENTERED AT 11:28:11 ON 17 JUL 2002)

FILE 'MEDLINE' ENTERED AT 11:28:44 ON 17 JUL 2002
L1 6 S LYSOSTAPHIN (2A) RESISTANT
L2 13 S (LYSOSTAPHIN (2A) (RESIST? OR INSENSITIVE)) NOT L1

FILE 'CAPLUS' ENTERED AT 11:35:50 ON 17 JUL 2002
L3 21 S L2 NOT L1

FILE 'MEDLINE, CAPLUS' ENTERED AT 11:36:15 ON 17 JUL 2002
L4 23 DUPLICATE REMOVE L2 L3 (11 DUPLICATES REMOVED)

=> d bib,abs 1-23

L4 ANSWER 1 OF 23 CAPLUS COPYRIGHT 2002 ACS
AN 2001:828921 CAPLUS
DN 135:352833
TI Topical lysostaphin therapy for Staphylococcus ocular infections
IN O'callaghan, Richard J.
PA Board of Supervisors of Louisiana State University and Agricultural and Mechanical College, USA
SO U.S., 6 pp.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6315996	B1	20011113	US 1999-289684	19990409

AB A method has been discovered for using lysostaphin as an effective antibiotic for topical treatment of Staphylococcus corneal infections (keratitis). Lysostaphin applied topically to the cornea by eye drops killed bacteria within the cornea; lysostaphin reduced the no. of bacteria from approx. 10,000,000 viable bacteria colony forming units ("CFU") in the untreated eye to essentially no viable bacteria in the treated eyes. Treatment by lysostaphin was more potent than any of the smaller antibiotics that have been previously tested (e.g., tetracyclines, erythromycin, cephalosporins, vancomycin, aminoglycosides, or fluoroquinolones). Moreover, topical application of lysostaphin was effective against the highly antibiotic-resistant Staphylococcus strains.
RE.CNT 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 2 OF 23 MEDLINE
AN 2001572049 MEDLINE
DN 21536883 PubMed ID: 11679550
TI Development of vancomycin and **lysostaphin resistance** in a methicillin-resistant Staphylococcus aureus isolate.
AU Boyle-Vavra S; Carey R B; Daum R S
CS Department of Pediatrics, University of Chicago, Chicago, IL, USA.. sboyleva@midway.uchicago.edu
NC RO1 AI 40481-01 BM (NIAID)
RO3 AI 44999-02 (NIAID)
SO JOURNAL OF ANTIMICROBIAL CHEMOTHERAPY, (2001 Nov) 48 (5) 617-25.
Journal code: 7513617. ISSN: 0305-7453.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200202
ED Entered STN: 20011029
Last Updated on STN: 20020221

Entered Medline: 20020220

AB Glycopeptide resistance in Staphylococcus aureus is poorly understood. The diversity of change documented in cell walls of clinical glycopeptide-intermediate S. aureus (GISA) isolates is evidence that a single genetic or biochemical change cannot account for resistance in all isolates described to date. Therefore, identification of new GISA clinical isolates provides an opportunity to gain insight into the range of adaptive strategies employed by staphylococci to survive in the presence of glycopeptides. In April 1999, a GISA isolate was obtained from the blood of a 63-year-old dialysis patient in Illinois. This isolate was one of six clonally identical MRSA isolates (A-F) serially obtained from the blood of this patient who was receiving vancomycin therapy. All isolates were resistant to oxacillin (MIC > 256 mg/L). The initial isolate had an MIC of vancomycin of 1 mg/L. However, the presence of a subpopulation that could grow in the presence of 5 mg/L of vancomycin indicated that this isolate was predisposed to the acquisition of the GISA phenotype (MIC of vancomycin 10-12 mg/L), which occurred 13 days later, associated with an increased MIC of the endopeptidase lysostaphin and slightly increased cell wall thickness. The first and last isolates in the series, A and F, resisted killing when incubated in vancomycin 2 mg/L, resisted autolysis when incubated in Triton X-100 and had a decreased expression of a c. 116 kDa autolytic band, properties that were different from glycopeptide-susceptible control isolates. **Lysostaphin resistance** was not accompanied by alterations in the peptidoglycan pentaglycine cross-bridge or a decrease in oxacillin MIC. These data, when taken together with the demonstration of increased cross-linking in isolate F compared with isolate A, demonstrate that vancomycin resistance in these isolates probably occurred by a mechanism different from that of other GISA isolates described to date.

L4 ANSWER 3 OF 23 CAPLUS COPYRIGHT 2002 ACS
AN 2001:37556 CAPLUS
DN 134:246913
TI Lysostaphin expression in mammary glands confers protection against staphylococcal infection in transgenic mice
AU Kerr, David E.; Plaut, Karen; Bramley, A. John; Williamson, Christine M.; Lax, Alistair J.; Moore, Karen; Wells, Kevin D.; Wall, Robert J.
CS Department of Animal Sciences, University of Vermont, Burlington, VT, 05405, USA
SO Nature Biotechnology (2001), 19(1), 66-70
CODEN: NABIF9; ISSN: 1087-0156
PB Nature America Inc.
DT Journal
LA English
AB Infection of the mammary gland, in addn. to causing animal distress, is a major economic burden of the dairy industry. Staphylococcus aureus is the major contagious mastitis pathogen, accounting for approx. 15-30% of infections, and has proved difficult to control using std. management practices. As a first step toward enhancing mastitis resistance of dairy animals, we report the generation of transgenic mice that secrete a potent anti-staphylococcal protein into milk. The protein, lysostaphin, is a peptidoglycan hydrolase normally produced by Staphylococcus simulans. When the native form is secreted by transfected eukaryotic cells it becomes glycosylated and inactive. However, removal of two glycosylation motifs through engineering asparagine to glutamine codon substitutions enables secretion of Gln125,232-lysostaphin, a bioactive variant. Three lines of transgenic mice, in which the 5'-flanking region of the ovine .beta.-lactoglobulin gene directed the secretion of Gln125,232-lysostaphin into milk, exhibit substantial resistance to an intramammary challenge of 104 colony-forming units (c.f.u.) of S. aureus, with the highest expressing line being completely resistant. Milk protein content and profiles of transgenic and nontransgenic mice are similar. These results clearly demonstrate the potential of genetic engineering to combat the most prevalent disease of dairy cattle.

RE.CNT 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 4 OF 23 CAPLUS COPYRIGHT 2002 ACS
AN 2000:161096 CAPLUS
DN 132:175811
TI A method using lysostaphin for the treatment of staphylococcal disease
IN Climo, Michael M.; Archer, Gordon L.; Goldstein, Beth P.
PA Ambi Inc., USA
SO PCT Int. Appl., 25 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000012049	A2	20000309	WO 1999-US20396	19990827
	WO 2000012049	A3	20000525		
	W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	US 6028051	A	20000222	US 1998-140732	19980827
	ZA 9905444	A	20000710	ZA 1999-5444	19990825
	AU 9958111	A1	20000321	AU 1999-58111	19990827
	EP 1107722	A2	20010620	EP 1999-945525	19990827
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	NO 2001000953	A	20010321	NO 2001-953	20010226
PRAI	US 1998-140732	A	19980827		
	US 1997-53470P	P	19970723		
	WO 1999-US20396	W	19990827		

AB Lysostaphin is an effective antibiotic in the treatment of staphylococcal infection. Large doses of lysostaphin or lysostaphin analogs are effective in short course, or even one-dose administration, in treating and eradicating staphylococcal infections, including those resistant to conventional antibiotics.

L4 ANSWER 5 OF 23 MEDLINE DUPLICATE 1
AN 1999177558 MEDLINE
DN 99177558 PubMed ID: 10077832
TI Identification of three additional femAB-like open reading frames in Staphylococcus aureus.
AU Tschierske M; Mori C; Rohrer S; Ehlert K; Shaw K J; Berger-Bachi B
CS Institute of Medical Microbiology, University of Zurich, Switzerland.
SO FEMS MICROBIOLOGY LETTERS, (1999 Feb 15) 171 (2) 97-102.
Journal code: 7705721. ISSN: 0378-1097.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AF106849; GENBANK-AF106850; GENBANK-AF106851
EM 199903
ED Entered STN: 19990413
Last Updated on STN: 19990413
Entered Medline: 19990329
AB Three new proteins, FmhA, FmhB and FmhC, with significant identities to FemA and FemB were identified in the Staphylococcus aureus (ATCC 55748) genome database. They were mapped to the SmaI-C, SmaI-H and SmaI-A

fragments of the S. aureus 8325 chromosome, respectively. Whereas insertional inactivation of fmhA and fmhC had no effects on growth, antibiotic susceptibility, **lysostaphin resistance**, or peptidoglycan composition of the strains, fmhB could not be inactivated, strongly suggesting that fmhB may be an essential gene. As deduced from the functions of FemA and FemB which are involved in the synthesis of the peptidoglycan pentaglycine interpeptide, FmhB may be a candidate for the postulated FemX thought to add the first glycine to the nascent interpeptide.

L4 ANSWER 6 OF 23 MEDLINE DUPLICATE 2
AN 1998295009 MEDLINE
DN 98295009 PubMed ID: 9631548
TI Zoocin A immunity factor: a femA-like gene found in a group C streptococcus.
AU Beatson S A; Sloan G L; Simmonds R S
CS Department of Microbiology, University of Otago, Dunedin, New Zealand.
SO FEMS MICROBIOLOGY LETTERS, (1998 Jun 1) 163 (1) 73-7.
Journal code: 7705721. ISSN: 0378-1097.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-U50357
EM 199807
ED Entered STN: 19980716
Last Updated on STN: 19980716
Entered Medline: 19980709
AB A 6.8-kb fragment of Streptococcus equi subsp. zooepidemicus 4881 DNA containing the zoocin A gene (zooA) was cloned in Escherichia coli and sequenced. We have identified a gene we call zoocin A immunity factor (zif), which protects the producer cell from the otherwise lethal action of its own product. Transformation of Streptococcus gordonii DL1 with zooA and zif changed its phenotypic character from a non-zoocin A producing-zoocin A sensitive cell to a zoocin A producing-zoocin A resistant cell. zif has sequence homology to femA (factor essential for methicillin **resistance**) and lif (**lysostaphin** immunity factor). No differences were observed in amino acid or amino sugar compositions of peptidoglycan purified from zoocin A sensitive vs. zoocin A immune cells.

L4 ANSWER 7 OF 23 MEDLINE DUPLICATE 3
AN 1999132637 MEDLINE
DN 99132637 PubMed ID: 9931440
TI Identification and molecular characterization of a gene homologous to epr (endopeptidase resistance gene) in Staphylococcus aureus.
AU Sugai M; Fujiwara T; Komatsuzawa H; SuginakaH
CS Department of Microbiology, Hiroshima University School of Dentistry, Hiroshima 734-8553,. Japan.sugai@ipc.hiroshima-u.ac.jp
SO GENE, (1998 Dec 11) 224 (1-2) 67-75.
Journal code: 7706761. ISSN: 0378-1119.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AB015195
EM 199902
ED Entered STN: 19990301
Last Updated on STN: 20000303
Entered Medline: 19990218
AB Certain Staphylococci possess a gene called epr or lif that renders the cells resistant to lysis by glycylglycine endopeptidase. The resistance is conferred by modifying the amino acid composition of interpeptide chains in cell-wall peptidoglycan by increasing serine content and decreasing

glycine content. A gene homologous to epr/lif was cloned from S. aureus RN450 genomic libraries and designated eprh. eprh was found to localize 27bp downstream of a novel cell-wall hydrolase gene lytN, which is in the same orientation with eprh. By analogy with epr/lif, eprh is suggested to be involved in the transfer of certain amino acids, possibly serine or amino acids other than glycine, to interpeptide chains of cell-wall peptidoglycan. Unlike epr/lif, overexpression of eprh in S. aureus did not result in an increased **resistance** to **lysostaphin**.
Insertional inactivation of eprh or lytN by Campbell-type integration did not affect the susceptibility of the cells to lysostaphin, either. These results suggest that eprh and lytN are not essential genes for S. aureus growth. The physiological function of eprh remains unknown.

L4 ANSWER 8 OF 23 CAPLUS COPYRIGHT 2002 ACS
AN 1998:346426 CAPLUS
DN 129:25648
TI Epr-specified **lysostaphin** endopeptidase **resistance**
AU Dehart, Heather Morton Posey
CS Univ. of Alabama, Tuscaloosa, AL, USA
SO (1997) 66 pp. Avail.: UMI, Order No. DA9821529
From: Diss. Abstr. Int., B 1998, 59(1), 63
DT Dissertation
LA English
AB Unavailable

L4 ANSWER 9 OF 23 MEDLINE DUPLICATE 4
AN 1998053874 MEDLINE
DN 98053874 PubMed ID: 9393725
TI Specificities of FemA and FemB for different glycine residues: FemB cannot substitute for FemA in staphylococcal peptidoglycan pentaglycine side chain formation.
AU Ehlert K; Schroder W; Labischinski H
CS PH-Research Antiinfectives I, Bayer AG, Wuppertal, Germany.
SO JOURNAL OF BACTERIOLOGY, (1997 Dec) 179 (23) 7573-6.
Journal code: 2985120R. ISSN: 0021-9193.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199712
ED Entered STN: 19980116
Last Updated on STN: 19980116
Entered Medline: 19971230
AB The femAB operon codes for two nearly identical approximately 50-kDa proteins involved in the formation of the staphylococcal pentaglycine interpeptide bridge. Sequencing and analysis of the femA region of mutants isolated by chemical mutagenesis and selection for **lysostaphin resistance** revealed point mutations leading to the expression of truncated FemA proteins. These femA mutants, although still producing an intact FemB, exhibited a phenotype identical as that described for femAB double mutants. Thus, FemA seems to be essential for the addition of glycine residues 2 and 3 only, whereas FemB is involved in the attachment of exclusively glycine residues 4 and 5. Although FemB has 39% identity with FemA, it cannot substitute for FemA. The FemA and FemB proteins seem to be highly specific in regard to the position of the glycine residues that they attach.

L4 ANSWER 10 OF 23 MEDLINE DUPLICATE 5
AN 97352690 MEDLINE
DN 97352690 PubMed ID: 9209049
TI epr, which encodes glycylglycine endopeptidase resistance, is homologous to femAB and affects serine content of peptidoglycan cross bridges in Staphylococcus capitis and Staphylococcus aureus.
AU Sugai M; Fujiwara T; Ohta K; Komatsuzawa H; Ohara M; Suginaka H

CS Department of Microbiology, Hiroshima University School of Dentistry, Minami-ku, Japan.. sugai@ipc.hiroshima-u.ac.jp
SO JOURNAL OF BACTERIOLOGY, (1997 Jul) 179 (13) 4311-8.
Journal code: 2985120R. ISSN: 0021-9193.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AB000222
EM 199708
ED Entered STN: 19970902
Last Updated on STN: 20000303
Entered Medline: 19970819
AB Staphylococcus capitis EPK1 produces a glycylglycine endopeptidase, ALE-1 (M. Sugai, T. Fujiwara, T. Akiyama, M. Ohara, H. Komatsuzawa, S. Inoue, and H. Suginaka, J. Bacteriol. 179:1193-1202, 1997), which hydrolyzes interpeptide pentaglycine chains of cell wall peptidoglycan of S. aureus. Characterizations of the enzyme activity and cloning of ale-1 revealed that ALE-1 is very similar to prolysostaphin produced by S. simulans bv. staphylolyticus. Strain EPK1 is resistant to lysis by ALE-1 and by lysostaphin. A gene that renders the cells resistant to glycylglycine endopeptidase (epr) was found 322 bp upstream of and in the opposite orientation to ale-1. The deduced amino acid sequence of epr showed similarities to FemA and FemB, which have been characterized as factors essential for methicillin resistance of S. aureus. Inactivation of either femA or femB causes decreased resistance to methicillin, increased **resistance** to **lysostaphin**, and decreased glycine content in the interpeptide chains of peptidoglycan. Therefore, femAB is suggested to be involved in the addition of glycine to pentapeptide peptidoglycan precursor. S. aureus with epr on a multicopy plasmid had phenotypes similar to those of femAB mutants except that it did not alter resistance level to methicillin. These results suggest that epr and femAB belong to the protein family involved in adding amino acids to the pentapeptide peptidoglycan precursor and that epr is involved in the addition of serine to the pentapeptide.

L4 ANSWER 11 OF 23 MEDLINE DUPLICATE 6
AN 97400268 MEDLINE
DN 97400268 PubMed ID: 9257762
TI Increased production of penicillin-binding protein 2, increased detection of other penicillin-binding proteins, and decreased coagulase activity associated with glycopeptide resistance in Staphylococcus aureus.
AU Moreira B; Boyle-Vavra S; deJonge B L; Daum R S
CS Department of Pediatrics, The University of Chicago, Illinois 60637, USA.
SO ANTIMICROBIAL AGENTS AND CHEMOTHERAPY, (1997 Aug) 41 (8) 1788-93.
Journal code: 0315061. ISSN: 0066-4804.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199709
ED Entered STN: 19971008
Last Updated on STN: 19980206
Entered Medline: 19970925
AB The mechanism of glycopeptide resistance in the genus Staphylococcus is unknown. Since these antimicrobial compounds act by binding the peptidoglycan precursor terminus, the target of transglycosylase and transpeptidase enzymes, it was hypothesized that resistance might be mediated in Staphylococcus aureus by increased production or activity of these enzymes, commonly called penicillin-binding proteins (PBPs). To evaluate this possibility, glycopeptide-resistant mutants were prepared by passage of several clinical isolates of this species in nutrient broth containing successively increasing concentrations of the glycopeptide vancomycin or teicoplanin. Decreased coagulase activity and increased

resistance to **lysostaphin** were uniformly present in the vancomycin-resistant mutants. Peptidoglycan cross-linking increased in one resistant isolate and decreased in two resistant isolates. The amounts of radioactive penicillin that bound to each PBP in susceptible and resistant strains were compared; PBP2 production was also evaluated by Western blotting. Increased penicillin labeling and production of PBP2 were found in all resistant derivatives selected by either vancomycin or teicoplanin. Moreover, the increase in PBP2 penicillin labeling occurred early in a series of vancomycin-selected derivatives and was strongly correlated ($r > 0.9$) with the increase in vancomycin and teicoplanin MIC. An increase in penicillin labeling also occurred, variably, in PBP1, PBP3, and/or PBP4. These data demonstrate a strong correlation between resistance to glycopeptides and increased PBP activity and/or production in S. aureus. Such an increase could allow PBPs to better compete with glycopeptides for the peptidoglycan precursor.

L4 ANSWER 12 OF 23 MEDLINE DUPLICATE 7
AN 97417793 MEDLINE
DN 97417793 PubMed ID: 9271851
TI Lif, the lysostaphin immunity factor, complements FemB in staphylococcal peptidoglycan interpeptide bridge formation.
AU Tschierske M; Ehlert K; Stranden A M; Berger-Bachi B
CS Institute of Medical Microbiology, University of Zurich, Switzerland.
SO FEMS MICROBIOLOGY LETTERS, (1997 Aug 15) 153 (2) 261-4.
Journal code: 7705721. ISSN: 0378-1097.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199709
ED Entered STN: 19971008
Last Updated on STN: 20000303
Entered Medline: 19970924
AB The formation of the Staphylococcus aureus peptidoglycan pentaglycine interpeptide chain needs FemA and FemB for the incorporation of glycines Gly2-Gly3, and Gly4-Gly5, respectively. The lysostaphin immunity factor Lif was able to complement FemB, as could be shown by serine incorporation and by an increase in **lysostaphin resistance** in the wild-type as well as in a femB mutant. However, Lif could not substitute for FemA in femA or in femAB-null mutants. Methicillin resistance, which is dependent on functional FemA and FemB, was not complemented by Lif, suggesting that serine-substituted side chains are a lesser substrate for penicillin-binding protein PBP2' in methicillin resistance.

L4 ANSWER 13 OF 23 MEDLINE DUPLICATE 8
AN 97136597 MEDLINE
DN 97136597 PubMed ID: 8981974
TI Cell wall monoglycine cross-bridges and methicillin hypersusceptibility in a femAB null mutant of methicillin-resistant Staphylococcus aureus.
AU Stranden A M; Ehlert K; Labischinski H; Berger-Bachi B
CS Institute of Medical Microbiology, University of Zurich, Switzerland.
SO JOURNAL OF BACTERIOLOGY, (1997 Jan) 179 (1) 9-16.
Journal code: 2985120R. ISSN: 0021-9193.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199702
ED Entered STN: 19970219
Last Updated on STN: 20000303
Entered Medline: 19970206
AB The femAB operon is involved in the formation of the characteristic pentaglycine side chain of the staphylococcal peptidoglycan. Allele replacement of the femAB operon with the tetracycline resistance

determinant tetK in a methicillin-resistant Staphylococcus aureus strain resulted in impaired growth, methicillin hypersusceptibility, and **lysostaphin resistance**. The usual pentaglycine cross-bridges were replaced by monoglycine bridges exclusively, and cross-linking of the peptidoglycan strands was drastically reduced. Complementation of the femAB null mutant by either femA or femAB resulted in the extension of the cross-bridges to a triglycine or a pentaglycine, respectively. This finding suggests that FemA is responsible for the formation of glycines 2 and 3, and FemB is responsible for formation of glycines 4 and 5, of the pentaglycine side chain of the peptidoglycan precursor. Moreover, it can be deduced that addition of the first glycine must occur by a femAB-independent mechanism.

L4 ANSWER 14 OF 23 MEDLINE DUPLICATE 9
AN 97302473 MEDLINE
DN 97302473 PubMed ID: 9158720
TI Staphylococcal peptidoglycan interpeptide bridge biosynthesis: a novel antistaphylococcal target?.
AU Kopp U; Roos M; Wecke J; Labischinski H
CS Bayer AG, Pharma Research Antiinfectives I, Wuppertal, Germany.
SO MICROBIAL DRUG RESISTANCE, (1996 Spring) 2 (1) 29-41.
Journal code: 9508567. ISSN: 1076-6294.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199706
ED Entered STN: 19970630
Last Updated on STN: 19970630
Entered Medline: 19970619
AB In staphylococci, crosslinking of the peptide moiety of peptidoglycan is mediated via an additional spacer, the interpeptide bridge, consisting of five glycine residues. The femAB operon, coding for two approximately 50-kDa proteins is known to be involved in pentaglycine bridge formation. Using chemical mutagenesis of the beta-lactam-resistant strain BB270 and genetic, biochemical, and biophysical characterization of mutants selected for loss of beta-lactam **resistance** and reduced **lysostaphin** sensitivity it is shown that peptide bridge formation proceeds via three intermediate bridge lengths (cell wall peptides with no, one, three, and five glycine units). To proceed from one intermediate to the next, three genes appear necessary: femX, femA, and femB. The drastic loss of beta-lactam resistance after inactivation of FemA or partial impairment of FemX even beyond the level of the sensitive wild-type strains renders these proteins attractive antistaphylococcal targets.

L4 ANSWER 15 OF 23 CAPLUS COPYRIGHT 2002 ACS
AN 1995:665834 CAPLUS
TI The **lysostaphin** endopeptidase **resistance** gene (epr) specifies modification of peptidoglycan cross bridges in Staphylococcus simulans and Staphylococcus aureus
AU DeHart, Heather Posey; Heath, Harry E.; Heath, Lucie S.; LeBlanc, Paul A.; Sloan, Gary L.
SO Appl. Environ. Microbiol. (1995), 61(7), 2811
CODEN: AEMIDF; ISSN: 0099-2240
DT Journal; Errata
LA English
AB Unavailable

L4 ANSWER 16 OF 23 MEDLINE DUPLICATE 10
AN 95266829 MEDLINE
DN 95266829 PubMed ID: 7747966
TI The **lysostaphin** endopeptidase **resistance** gene (epr) specifies modification of peptidoglycan cross bridges in Staphylococcus

simulans and Staphylococcus aureus.
CM Erratum in: Appl Environ Microbiol 1995 Jul;61(7):2811
AU DeHart H P; Heath H E; Heath L S; LeBlanc P A; Sloan G L
CS Department of Biological Sciences, University of Alabama, Tuscaloosa 35487, USA.
SO APPLIED AND ENVIRONMENTAL MICROBIOLOGY, (1995 Apr) 61 (4) 1475-9. Journal code: 7605801. ISSN: 0099-2240.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199506
ED Entered STN: 19950621
Last Updated on STN: 20000303
Entered Medline: 19950612
AB Staphylococcus simulans biovar staphylolyticus produces an extracellular glycylglycine endopeptidase (lysostaphin) that lyses other staphylococci by hydrolyzing the cross bridges in their cell wall peptidoglycans. The genes for endopeptidase (end) and endopeptidase resistance (epr) reside on plasmid pACK1. An 8.4-kb fragment containing end was cloned into shuttle vector pL150 and was then introduced into Staphylococcus aureus RN4220. The recombinant S. aureus cells produced endopeptidase and were resistant to lysis by the enzyme, which indicated that the cloned fragment also contained epr. Treatments to remove accessory wall polymers (proteins, teichoic acids, and lipoteichoic acids) did not change the endopeptidase sensitivity of walls from strains of S. simulans biovar staphylolyticus or of S. aureus with and without epr. Immunological analyses of various wall fractions showed that there were epitopes associated with endopeptidase resistance and that these epitopes were found only on the peptidoglycans of epr+ strains of both species. Treatment of purified peptidoglycans with endopeptidase confirmed that resistance or susceptibility of both species was a property of the peptidoglycan itself. A comparison of the chemical compositions of these peptidoglycans revealed that cross bridges in the epr+ cells contained more serine and fewer glycine residues than those of cells without epr. The presence of the 8.4-kb fragment from pACK1 also increased the susceptibility of both species to methicillin.

L4 ANSWER 17 OF 23 CAPLUS COPYRIGHT 2002 ACS
AN 1991:486327 CAPLUS
DN 115:86327
TI femA, which encodes a factor essential for expression of methicillin resistance, affects glycine content of peptidoglycan in methicillin-resistant and methicillin-susceptible Staphylococcus aureus strains
AU Maidhof, Heinrich; Reinicke, Bernhard; Bluemel, Peter; Berger-Baechi, Brigitte; Labischinski, Harald
CS Robert Koch-Inst., Fed. Health Off., Berlin, D-1000/65, Fed. Rep. Ger.
SO J. Bacteriol. (1991), 173(11), 3507-13
CODEN: JOBAAY; ISSN: 0021-9193
DT Journal
LA English
AB femA Is a chromosomally encoded factor, occurring naturally in S. aureus, which is essentially for the expression of high-level methicillin resistance in this organism. The prodn. of a low-affinity penicillin-binding protein PBP2a or PBP2' which is intimately involved with methicillin resistance in S. aureus, is not influenced by femA. To elucidate a possible physiol. function of the 48-kDa protein encoded by femA, several related methicillin-resistant, methicillin-susceptible, and Tn551 insertionally inactivated femA mutants were analyzed for possible changes in cell wall structure and metab. Independent of the presence of mec, the methicillin-resistance determinant, all femA mutants had a reduced peptidoglycan (PG) glycine content (up to 60% in the molar ratio of glycine/glutamic acid) compared to that of related femA+ parent strains. Addnl. effects of femA inactivation and the subsequent decrease

in PG-assocd. glycine were (1) reduced digestion of PG by recombinant lysostaphin, (2) unaltered digestion of PG by Chalaropsis B-muramidase, (3) reduced cell wall turnover, (4) reduced whole-cell autolysis, and (5) increased sensitivity towards B-lactam antibiotics. Also, the PG-assocd. glycine content of a femA::Tn551 methicillin-susceptible strain was restored concomitantly with the methicillin resistance to a level almost equal to that of its femA+ methicillin-resistant parent strain by introduction of plasmid pBBB31, encoding femA.

L4 ANSWER 18 OF 23 MEDLINE DUPLICATE 11
AN 89273565 MEDLINE
DN 89273565 PubMed ID: 2730641
TI Plasmid-encoded **lysostaphin** endopeptidase **resistance** of Staphylococcus simulans biovar staphylolyticus.
AU Heath H E; Heath L S; Nitterauer J D; Rose K E; Sloan G L
CS Department of Microbiology, University of Alabama, Tuscaloosa 35487.
SO BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1989 May 15) 160 (3) 1106-9.
Journal code: 0372516. ISSN: 0006-291X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198907
ED Entered STN: 19900309
Last Updated on STN: 20000303
Entered Medline: 19890710
AB Staphylococcus simulans biovar staphylolyticus, the lysostaphin-producing organism, secretes a staphylolytic endopeptidase (EC 3.4.99.17) that is encoded on plasmid pACK1. Susceptibility of pACK1-cured strains to lysis by endopeptidase established that resistance to this enzyme is not an inherent property of the organism but rather is encoded on this dispensable plasmid. Furthermore, the enzyme is not an autolysin that is essential for cell wall synthesis because strains lacking the endopeptidase gene grew normally.

L4 ANSWER 19 OF 23 MEDLINE
AN 76025376 MEDLINE
DN 76025376 PubMed ID: 1176603
TI Virulence factors of biotypes of Staphylococcus epidermidis from clinical sources.
AU Males B M; Rogers W A Jr; Parisi J T
SO JOURNAL OF CLINICAL MICROBIOLOGY, (1975 Mar) 1 (3) 256-61.
Journal code: 7505564. ISSN: 0095-1137.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 197512
ED Entered STN: 19900313
Last Updated on STN: 20000303
Entered Medline: 19751230
AB The biotyping scheme of Baird-Parker was applied to cultures of Staphylococcus epidermidis from patients. In all, 63.6% of 228 cultures belonged to biotype 1, followed by biotypes 4, 3, and 2 in decreasing order of incidence. When classified according to clinical source of isolation, cultures of S. epidermidis were most frequently isolated from urine, with 39.5% of 228 cultures from this source. Each of the four biotypes was distributed throughout all nine catagories of clinical sources. The production of virulence factors was based on the results of three groups of tests: (i) deoxyribonuclease, urease, gelatinase, caseinase, and lysozyme production; (ii) lipolytic activity on the tweens; and (iii) hemolysin production. Enzymatic activity was highest for organisms in biotypes 1, followed by biotypes 3, 4, and 2 in decreasing

order. Of the 228 cultures, 76.3% were lysed by **lysostaphin**. **Resistance** to antibiotics was highest for tetracycline, ampicillin, and penicillin, with rates of 54.8, 69.3, and 81.6%, respectively. The role of S. epidermidis as an etiological agent was studied by analyzing the laboratory and clinical data of 80 patients selected at random with bacteriuric S. epidermidis. Organisms in biotype 1 were most commonly associated with urinary tract infection. The significance of certain biotypes of S. epidermidis as opportunistic pathogens among compromised hosts in a hospital environment is discussed.

L4 ANSWER 20 OF 23 CAPLUS COPYRIGHT 2002 ACS
AN 1974:563936 CAPLUS
DN 81:163936
TI Lack of correlation between methicillin resistance and susceptibility of lysostaphin in Staphylococcus aureus
AU Chopra, I.; Lacey, R. W.
CS Med. Sch., Univ. Bristol, Bristol, UK
SO J. Gen. Microbiol. (1974), 82, Pt. 2, 419-20
CODEN: JGMIAN
DT Journal
LA English
AB The acquisition of methicillin [61-32-5] resistance by Stephylococcus aureus did not affect its susceptibility to lysis by lysostaphin. This was illustrated by comparing the rates of lysis by lysostaphin in pairs of strains that differed only by the possession of gene(s) for methicillin resistance.

L4 ANSWER 21 OF 23 CAPLUS COPYRIGHT 2002 ACS
AN 1968:425280 CAPLUS
DN 69:25280
TI Lysostaphin. I. Sensitivity of 355 Staphylococcus aureus strains of human origin to lysostaphin
AU Pulverer, G.
CS Univ. Koeln, Cologne, Ger.
SO Z. Med. Mikrobiol. Immunol. (1968), 154(1), 40-8
CODEN: ZMMIA3
DT Journal
LA German
AB The effect of lysostaphin at concns. between 5.0 and 0.037 .mu.g./ml. on 355 coagulase-pos. S. aureus strains of human origin in Difco medium no. 3 and an exposure time of 24 hrs. at 37.degree. was investigated. All strains were resistant to penicillin G (1.5 units), streptomycin (25 .mu.g.), chloramphenicol (10 .mu.g.), tetracycline (10 .mu.g.), and erythromycin (10 .mu.g.), and 39 were resistant to methicillin. Of the remainder, there were 300 nonepidemic and 16 epidemic strains. At a min. inhibitory concn. of 2.5 .mu.g./ml., 1.25 .mu.g./ml., 0.62 .mu.g./ml., 0.31 .mu.g./ml., 0.15 .mu.g./ml., 0.075 .mu.g./ml. and 0.037 .mu.g./ml. there were 8, 44, 101, 103, 74, 23, and 2 strains affected, resp., while all strains were killed at a concn. of 5.0 .mu.g./ml. The mode of action of lysostaphin was not related to that of methicillin, since methicillin resistance was not correlated with **lysostaphin resistance**. There was also no correlation between lysostaphin sensitivity and phage group or egg yolk activity.

L4 ANSWER 22 OF 23 CAPLUS COPYRIGHT 2002 ACS
AN 1967:113232 CAPLUS
DN 66:113232
TI Lysostaphin: an enzymic approach to staphylococcal disease. I. In vitro studies
AU Schaffner, William; Melly, M. Ann; Hash, John H.; Koenig, M. Glenn
CS Sch. of Med., Vanderbilt Univ., Nashville, Tenn., USA
SO Yale J. Biol. Med. (1967), 39(4), 215-29
CODEN: YJBMAU
DT Journal

LA English
AB Lysostaphin (I) (70 .gamma./ml.) rapidly killed Staphylococcus aureus in vitro, unlike the penicillins. I maintained its activity in serum and after heating at 37.degree.. I was equally active against encapsulated and nonencapsulated staphylococcal strains; like penicillin, I was inactive against intracellular staphylococci. I-resistant staphylococci were isolated in vitro; these differed from the parent strain in colonial morphology, slower GROWTH RATES, increased penicillin sensitivity, and occasionally in diminished virulence for mice. Resistance may possibly relate to configurattional alterations on the cell wall surface rather than to changes in its chem. composition. 24 references.

L4 ANSWER 23 OF 23 CAPLUS COPYRIGHT 2002 ACS
AN 1967:420242 CAPLUS
DN 67:20242
TI Experimental observations on staphylococcal disease
AU Rogers, David Elliott
CS Vanderbilt Univ., Nashville, Tenn., USA
SO Postepy Mikrobiol. (1965), 5(2), 279-96
CODEN: PMKMAV
DT Journal
LA English
AB This report points out the nature of the factors conferring staphylococci virulence, the reasons for the refractoriness of staphylococcal infections to treatment, and the role of humoral immunity in prevention of staphylococcal disease in man. Coagulase-pos. staphylococci isolated from human disease could be grown from human granulocytes after 4-5 hrs. of intracellular residence. Coagulase-neg. strains were rapidly destroyed under these circumstances. The survival of part of the microbial populations within the cells is an important attribute of virulent strains. When a penicillinsensitive staphylococcus is incorporated within human leukocytes the lethal effects of penicillin are blocked. Similar results were obtained using lysostaphin which is highly active against all strains of staphylococci tested. It was demonstrated that levels of bacteremia can be modified by manipulating leukocyte levels. Studies were reported of a strain of Staphylococcus which dissocd. into 2 colonial variants when incorporated in soft agar contg. plasma. One variant was virulent for mice when injected i.p. and grew in diffuse colonies. The other variant was virulent under similar conditions and grew in compact colonies. The diffuse variant was found to have a surface component which prevents phagocytosis. Studies with 12 different mouse virulent staphylococcal strains show that all have an antigenic capsule and require other factors for ingestion by leukocytes. Penicillin was without effect on staphylococci at population titers similar to those contained in abscesses. High titers and relatively sluggish metabolism of organisms in the abscess cavity may have an important bearing on the refractoriness of lesions to antimicrobial therapy.

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=> s lysostaphin (2a) resistant
          366 LYSOSTAPHIN
       135390 RESISTANT
          150 RESISTANTS
       135412 RESISTANT
                 (RESISTANT OR RESISTANTS)
L1           6 LYSOSTAPHIN (2A) RESISTANT

=> d bib,abs 1-6
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L1 ANSWER 1 OF 6 MEDLINE
AN 2002293213 IN-PROCESS
DN 22013727 PubMed ID: 12019130
TI Combinations of lysostaphin with beta-lactams are synergistic against oxacillin-resistant Staphylococcus epidermidis.
AU Kiri Nandini; Archer Gordon; Climo Michael W
CS Department of Medicine, Virginia Commonwealth University Health Science System, Richmond, Virginia 23249, USA.
NC R-41HL60334 (NHLBI)
SO ANTIMICROBIAL AGENTS AND CHEMOTHERAPY, (2002 Jun) 46 (6) 2017-20. Journal code: 0315061. ISSN: 0066-4804.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS IN-PROCESS; NONINDEXED; Priority Journals
ED Entered STN: 20020530
Last Updated on STN: 20020530
AB Oxacillin-resistant Staphylococcus aureus is rapidly killed by the endopeptidase lysostaphin, and the addition of beta-lactam antibiotics provides synergistic killing. We investigated the possibility that beta-lactams given in combination with lysostaphin would improve the activity of **lysostaphin** against oxacillin-**resistant** Staphylococcus epidermidis (ORSE), which is normally less susceptible to lysostaphin. Checkerboard synergy testing was performed for lysostaphin given in combination with oxacillin against 10 ORSE isolates for which the lysostaphin MICs were > o r= 8 microg/ml. The fractional inhibitory concentration index ranged from 0.0234 to 0.2656, indicating synergy, which was confirmed in growth curve experiments. In the rabbit model of experimental aortic valve endocarditis using an ORSE strain, the combination of lysostaphin and nafcillin was as effective as vancomycin alone and significantly better than lysostaphin or nafcillin alone. We conclude that beta-lactam antibiotics given in combination with lysostaphin are synergistic against many strains of ORSE.

L1 ANSWER 2 OF 6 MEDLINE
AN 2001435424 MEDLINE
DN 21199336 PubMed ID: 11302806
TI Mechanism and suppression of lysostaphin resistance in oxacillin-resistant Staphylococcus aureus.
AU Climo M W; Ehlert K; Archer G L
CS Department of Medicine, Hunter Holmes McGuire Veterans Affairs Medical Center, Richmond, Virginia 23249, USA.. Michael.Climo@med.va.gov

aureus mutant was successfully devised. Lysostaphin was sufficiently absorbed on the heat-killed mutant cells derived from S. aureus Cowan I and efficiently eluted by 3 M KSCN. Enzyme preparation obtained by a single procedure of the affinity purification was pure enough for practical use. The yield of the enzyme was 25 mg from 1 liter culture and recovery rate was 64%.

L1 ANSWER 4 OF 6 MEDLINE
AN 91072619 MEDLINE
DN 91072619 PubMed ID: 2254432
TI Effect of BiTek agar on lysostaphin susceptibility of staphylococci.
AU Langlois B E; Dawson K; Akers K
CS Department of Animal Sciences, University of Kentucky, Lexington 40546-0215.
SO JOURNAL OF CLINICAL MICROBIOLOGY, (1990 Nov) 28 (11) 2568-9. Journal code: 7505564. ISSN: 0095-1137.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199101
ED Entered STN: 19910308
Last Updated on STN: 20000303
Entered Medline: 19910124
AB Staphylococci which were considered to be lysostaphin susceptible on P agar containing Bacto-Agar showed different degrees of resistance to lysostaphin when tested on P agar made with BiTek agar. As a result, lysostaphin-susceptible strains were misidentified as **lysostaphin -resistant** strains.

L1 ANSWER 5 OF 6 MEDLINE
AN 86224542 MEDLINE
DN 86224542 PubMed ID: 3519667
TI Rapid lysostaphin test to differentiate Staphylococcus and Micrococcus species.
AU Geary C; Stevens M
SO JOURNAL OF CLINICAL MICROBIOLOGY, (1986 Jun) 23 (6) 1044-5. Journal code: 7505564. ISSN: 0095-1137.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198606
ED Entered STN: 19900321
Last Updated on STN: 20000303
Entered Medline: 19860627
AB A rapid, simple lysostaphin lysis susceptibility test to differentiate the genera Staphylococcus and Micrococcus was evaluated. Of 181 strains from culture collections, 95 of 95 Staphylococcus strains were lysed, and 79 of 79 Micrococcus strains were not lysed. The seven Planococcus strains were resistant. Clinical isolates (890) were tested with lysostaphin and for the ability to produce acid from glycerol in the presence of erythromycin. Overall agreement between the methods was 99.2%. All clinical Micrococcus strains (43) were **resistant** to **lysostaphin**, and all clinical Staphylococcus strains (847) were susceptible. Seven of the Staphylococcus strains did not produce acid from glycerol in the presence of erythromycin. This lysostaphin test provides results in 2 h. It is easier to perform than previously described lysostaphin lysis methods. It is also more rapid and accurate than the glycerol-erythromycin test.

L1 ANSWER 6 OF 6 MEDLINE
AN 77153627 MEDLINE
DN 77153627 PubMed ID: 848206
TI [Evaluation of phagocytosis of Staphylococcus aureus with the aid of

NC R-41HL60334 (NHLBI)
SO ANTIMICROBIAL AGENTS AND CHEMOTHERAPY, (2001 May) 45 (5) 1431-7.
Journal code: 0315061. ISSN: 0066-4804.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200108
ED Entered STN: 20010806
Last Updated on STN: 20010806
Entered Medline: 20010802
AB The potential for the development of resistance in oxacillin-resistant Staphylococcus aureus (ORSA) to lysostaphin, a glycylglycine endopeptidase produced by Staphylococcus simulans biovar staphylolyticus, was examined in vitro and in an in vivo model of infection. Following in vitro exposure of ORSA to subinhibitory concentrations of **lysostaphin**, **lysostaphin-resistant** mutants were idenitifed among all isolates examined. Resistance to lysostaphin was associated with a loss of resistance to beta-lactams and a change in the muropeptide interpeptide cross bridge from pentaglycine to a single glycine. Mutations in femA, the gene required for incorporation of the second and third glycines into the cross bridge, were found following PCR amplification and nucleotide sequence analysis. Complementation of **lysostaphin-resistant** mutants with pBBB31, which encodes femA, restored the phenotype of oxacillin resistance and lysostaphin susceptibility. Addition of beta-lactam antibiotics to lysostaphin in vitro prevented the development of **lysostaphin-resistant** mutants. In the rabbit model of experimental endocarditis, administration of a low dose of lysostaphin for 3 days led predictably to the appearance of **lysostaphin-resistant** ORSA mutants in vegetations. Coadministration of nafcillin with lysostaphin prevented the emergence of **lysostaphin-resistant** mutants and led to a mean reduction in aortic valve vegetation counts of 7.5 log(10) CFU/g compared to those for untreated controls and eliminated the isolation of **lysostaphin-resistant** mutants from aortic valve vegetations. Treatment with nafcillin and lysostaphin given alone led to mean reductions of 1.35 and 1.65 log(10) CFU/g respectively. In ORSA, resistance to lysostaphin was associated with mutations in femA, but resistance could be suppressed by the coadministration of beta-lactam antibiotics.

L1 ANSWER 3 OF 6 MEDLINE
AN 93233503 MEDLINE
DN 93233503 PubMed ID: 8474355
TI Efficient adsorption of lysostaphin on bacterial cells of **lysostaphin-resistant** Staphylococcus aureus mutant.
AU Sakurada J; Murai M; Zhijun L; Usui A; Seki K; Kobayashi K; Sumi Y; Jitsukawa H; Masuda S
CS Department of Bacteriology, Jikei University School of Medicine, Tokyo, Japan.
SO MICROBIOLOGY AND IMMUNOLOGY, (1993) 37 (1) 29-34.
Journal code: 7703966. ISSN: 0385-5600.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199305
ED Entered STN: 19930604
Last Updated on STN: 20000303
Entered Medline: 19930520
AB A simple and efficient method for the purification of staphylolytic endopeptidase (lysostaphin) contained in culture supernatant of Staphylococcus simulans biovar staphylolyticus strain by adsorption of the enzyme on bacterial cells of **lysostaphin-resistant** S.

lysostaphin (author's transl)].
Auswertung der Phagozytose von Staphylokokken unter Verwendung von Lysostaphin.

AU Dorner I; Blobel H; Schaeg W

SO ZENTRALBLATT FUR BAKTERIOLOGIE, PARASITENKUNDE, INFEKTIONSKRANKHEITEN UND HYGIENE. ERSTE ABTEILUNG ORIGINALE. REIHE A: MEDIZINISCHE MIKROBIOLOGIE UND PARASITOLOGIE, (1977) 237 (2-3) 141-6.
Journal code: 0331570. ISSN: 0300-9688.
CY GERMANY, WEST: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA German
FS Priority Journals
EM 197705
ED Entered STN: 19900313
Last Updated on STN: 19900313
Entered Medline: 19770520

AB The Staphylococcus aureus strains HV 1 and K 807 were lyzed by lysostaphin. S. epidermis E 1 and staphylococci extracted with guanidinium chloride were **resistant** to **lysostaphin**-induced lysis.
In the phagocytosis of S. aureus lysostaphin proved to be most useful for the differentiation between engulfed and extracellular staphylococci, particularly those attached to the surface of the polymorphonuclear granulocytes. It enabled a better recognition of the phagocytized staphylococci and therefore a more precise analysis of the phagocytosis experiments. A further improvement in the evaluation of phagocytosis was possible by the use of radioisotope labelling of staphylococci. This technique in combination with lysostaphin, might be useful for large-scale phagocytosis studies.